MATHEMATICAL MODELING OF THE IMPACT OF TREATMENT ON SCABIES CONTROL IN A POPULATION

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ABSTRACT

Scabies, caused by *Sarcoptes scabiei*, remains a significant public health concern, particularly in overcrowded and resource-limited settings where transmission is easily maintained. In this study, a deterministic SEITR compartmental model is formulated to describe the spread of scabies and to examine how treatment contributes to its management. The model analysis involves computing the basic reproduction number, establishing solution well-posedness, preserving the non-negativity of state variables, and characterizing the feasible invariant region. The infection-free and endemic steady states are obtained, and the local asymptotic stability of the infection-free equilibrium is investigated. In addition, a sensitivity analysis is conducted to identify the model factors with the greatest influence on transmission. The Homotopy Perturbation Method is subsequently employed to construct approximate solutions to the nonlinear system. The results indicate that effective treatment can markedly lower infection prevalence and improve the prospects for controlling scabies in the population.

1. INTRODUCTION

Scabies is a transmissible skin disease caused by the mite *Sarcoptes scabiei* var. *hominis*. The mite burrows into the outer layer of the skin, where it lays eggs and triggers a strong inflammatory response that often leads to intense itching, skin rashes, and irritation (World Health Organization, 2023; Mitchell *et al.*, 2024). It spreads mainly through prolonged skin-to-skin contact, although clothing, bedding, and other shared materials may also contribute to transmission, especially in severe cases with high mite density (World Health Organization, 2023; Centers for Disease Control and Prevention, 2024a; Uzun *et al.*, 2024). Scabies continues to be a serious health problem in many parts of the world, particularly in settings where overcrowding, poverty, poor access to healthcare, and close daily contact make transmission easier to maintain (World Health Organization, 2023; van der Linden *et al.*, 2019; Mitchell *et al.*, 2024). Current estimates indicate that more than 200 million people are affected at any given time, with children, older adults, and other vulnerable groups in tropical and low-income regions carrying much of the burden (World Health Organization, 2023). Beyond its visible skin symptoms, scabies can disrupt sleep, reduce quality of life, and affect social wellbeing. In some cases, it may also lead to secondary bacterial infections and serious complications such as invasive bacterial infection and rheumatic heart disease in endemic communities (World Health Organization, 2023; Iyengar *et al.*, 2024; Mitchell *et al.*, 2024).

Treatment remains one of the main approaches to scabies control because it helps affected individuals recover while also reducing the chance of further spread within the community (Centers for Disease Control and Prevention, 2024b; Uzun *et al*., 2024). Common treatment options include topical drugs such as permethrin, while oral ivermectin is often used in particular situations such as crusted scabies, institutional outbreaks, or cases that do not respond well to standard treatment (Centers for Disease Control and Prevention, 2024b; Iyengar *et al*., 2024; Uzun *et al*., 2024). Even so, controlling scabies in a population involves more than having effective drugs available. Good outcomes also depend on early diagnosis, proper use of medication, adherence to repeated treatment when required, simultaneous treatment of close contacts and household members, and appropriate handling of clothing, bedding, and other possible sources of re-exposure (Centers for Disease Control and Prevention, 2024b; Uzun *et al*., 2024). Reinfection remains a major difficulty, since untreated contacts, mild or unnoticed infections, and poor compliance with treatment instructions can allow transmission to continue within households and communities despite the availability of effective therapy (World Health Organization, 2023; Centers for Disease Control and Prevention, 2024b; Mitchell *et al*., 2024). For this reason, treatment should be viewed not only as care for infected individuals but also as an important factor in shaping transmission at the population level.

Mathematical modelling provides a useful way to understand how infectious diseases spread and to assess the likely impact of interventions such as treatment, isolation, vaccination, and public health education (Kermack and McKendrick, 1927; Hethcote, 2000). By dividing the population into epidemiological groups and describing movement between those groups with differential equations, such models make it possible to study long-term disease behaviour, equilibrium states, and threshold conditions for persistence or elimination (Kermack and McKendrick, 1927; Hethcote, 2000). Recent studies have continued to demonstrate the importance of mathematical modelling in investigating infectious disease dynamics and intervention effects across different epidemiological settings, including hepatitis B, Zika virus, and COVID-19 (Olaosebikan *et al*., 2025; Alaje *et al*., 2025; Ayoola *et al*., 2025). In the case of scabies, compartmental models are particularly helpful for exploring how treatment coverage, treatment effectiveness, reinfection, and contact patterns affect disease levels over time (van der Linden *et al*., 2019; Lydeamore *et al*., 2019). Although scabies causes substantial illness worldwide, mathematical studies on its transmission remain relatively limited when compared with many other infectious diseases. This gap makes further modelling work necessary, especially for understanding the role of treatment in reducing transmission (van der Linden *et al*., 2019). Because epidemiological models are often nonlinear, exact closed-form solutions are frequently difficult to obtain. Consequently, several approximate and semi-analytical methods have been developed for analysing nonlinear systems of differential equations arising in infectious disease modelling. Among these methods, the Homotopy Perturbation Method (HPM), originally introduced by He (1999), has remained a useful analytical technique for constructing approximate series solutions to nonlinear problems. An important advantage of HPM is that it does not require the presence of a naturally small parameter, which makes it suitable for epidemiological systems whose nonlinear terms do not readily fit within the framework of classical perturbation methods. Recent studies have further demonstrated the applicability of homotopy-based and semi-analytical techniques in infectious disease modelling, including applications to Zika virus and COVID-19 transmission models (Alaje *et al*., 2025; Ayoola *et al*., 2025). In addition, more recent developments of HPM for strongly nonlinear differential equations continue to support its effectiveness, convergence, and analytical flexibility (Roy & Maiti, 2023). For these reasons, HPM is adopted in this study to obtain approximate solutions to the proposed scabies model.

Motivated by the persistent global burden of scabies, the importance of treatment as a control strategy, and the value of mathematical analysis in public health research, this study formulates and analyzes a deterministic SEITR compartmental model for scabies transmission. The model consists of a nonlinear system of ordinary differential equations that describes the progression of individuals through five epidemiological compartments. The study derives the disease-free and endemic equilibria, computes the basic reproduction number, examines the local stability of the disease-free equilibrium, and evaluates the influence of key parameters through sensitivity analysis. Furthermore, the qualitative dynamics of the model are investigated, and the Homotopy Perturbation Method is applied to obtain semi-analytical approximate solutions and to numerically illustrate the effect of treatment on the exposed, infected, treated, and recovered populations over time. The main objective is to determine how treatment coverage and effectiveness can reduce disease prevalence, suppress transmission, and improve scabies control at the population level.

2 MATERIALS AND METHODS

2.1 Homotopy Perturbation Method

To obtain approximate analytical solutions of the nonlinear scabies transmission model, the Homotopy Perturbation Method (HPM) is employed. This method constructs a continuous deformation between a simpler auxiliary problem and the original nonlinear problem through an embedding parameter. As a semi-analytical technique, it is particularly useful for nonlinear systems in which exact closed-form solutions are difficult to obtain.

Consider the general nonlinear differential equation

$$\mathrm{D}(\varpi) - \mathrm{g}(\tau) = 0, \qquad \tau \in \Omega, \tag{1}$$

subject to the initial or boundary condition

$$\mathrm{B}\left(\varpi, \frac{\partial \varpi}{\partial n}\right) = 0, \qquad \tau \in \partial\Omega, \tag{2}$$

where D denotes a general nonlinear differential operator, B is the boundary operator, $\mathrm{g}(\tau)$ is a known source term, Ω is the solution domain, and $\varpi(\tau)$ is the unknown function to be determined.

The operator D is decomposed into linear and nonlinear parts as

$$\mathrm{D}(\varpi) = \mathrm{L}(\varpi) + \mathrm{N}\,(\varpi), \tag{3}$$

where L and N represent the linear and nonlinear operators, respectively.

Following the HPM procedure, a homotopy is constructed as

$$\mathrm{H}(\omega, \lambda) = (1-\lambda)\left[\mathrm{L}(\omega) - \mathrm{L}(\varpi_0)\right] + \lambda\left[\mathrm{D}(\omega) - \mathrm{g}(\tau)\right] = 0, \qquad \lambda \in [0,1], \tag{4}$$

where λ is the embedding parameter and ϖ_0 is an initial approximation satisfying the prescribed initial or boundary conditions.

When $\lambda = 0$, equation (4) reduces to

$$\mathrm{L}(\omega) - \mathrm{L}(\varpi_0) = 0, \tag{5}$$

which yields the simpler problem

$$\omega = \varpi_0. \tag{6}$$

When $\lambda = 1$, equation (4) becomes

$$\mathrm{D}(\omega) - \mathrm{g}(\tau) = 0, \tag{7}$$

which is the original nonlinear problem given in (1).

The solution is then expressed as a power series in λ:

$$\omega = \omega_0 + \lambda\omega_1 + \lambda^2\omega_2 + \lambda^3\omega_3 + \cdots. \tag{8}$$

Substituting equation (8) into equation (4) and equating the coefficients of like powers of λ produces a sequence of simpler subproblems that can be solved successively for $\omega_0, \omega_1, \omega_2, \ldots$. Letting $\lambda \to 1$ then gives the approximate solution of the original nonlinear problem:

$$\varpi = \lim_{\lambda \to 1} \omega = \omega_0 + \omega_1 + \omega_2 + \omega_3 + \cdots. \quad (9)$$

In the present study, this procedure is applied to the nonlinear SEITR scabies model. Accordingly, each state variable, namely $S(\tau)$, $E(\tau)$, $I(\tau)$, $T(\tau)$, and $R(\tau)$, is expanded in a homotopy series and solved recursively to obtain approximate analytical expressions for the model trajectories.

2.2 Existing Model

Fadhal *et al.* (2025) investigated scabies transmission dynamics by developing a delay differential equation model that classified the population into four groups: unvaccinated susceptibles, vaccinated susceptibles, infected individuals, and recovered individuals. The model accounted for time delays in disease progression and allowed for the re-entry of recovered individuals into infection-relevant pathways.

$$\left.\begin{aligned}
\frac{dS_u}{dt} &= \Lambda\rho S_v(t) - \frac{\beta i(t-\tau)}{N} S_u(t-\tau)e^{-\mu t} - (\mu+\alpha)S_u(t), t \geq 0 \\
\frac{dS_v}{dt} &= \alpha S_u(t) - \delta\frac{\beta I(t-\tau)}{N} S_v(t-\tau)e^{-\mu\tau} - (\mu+\rho)S_v(t), t \geq o \\
\frac{dI(t)}{dt} &= \frac{\beta I(t-\tau)}{N} S_u(t-\tau)e^{-\mu\tau} + \delta\frac{\beta I(t-\tau)}{N} S_v(t-\tau)e^{-\mu\tau} + \sigma\lambda R(t) - (\mu+\gamma)I(t), t \geq 0 \\
\frac{dR}{dt} &= \gamma I(t) - \sigma\lambda R(t) - \mu R(t), t \geq 0
\end{aligned}\right\}$$

2.3 Model Formulation

In this study, the total population $N(t)$ is subdivided into five compartments:

$S(t), E(t), I(t), T(t), R(t),$

where:

- $S(t)$ are those individuals within the population who have not yet been exposed and remain at risk of infection at time (t),
- $E(t)$ denotes the subset of the population that has been exposed to the scabies mite at time t but has not yet progressed to active infection,
- $I(t)$ represents the individuals who have developed an active scabies infestation and are capable of transmitting the mite to others at time (t),

• $T(t)$ refers to the portion of the population that is currently receiving therapeutic intervention for scabies infestation at time (t),

• $R(t)$ denotes the individuals who have successfully cleared the infestation following treatment and have transitioned to a recovered state at time (t).

Thus, $N(t) = S(t) + E(t) + I(t) + T(t) + R(t).$ (10)

The proposed SEITR scabies model results:

$$\frac{dS}{dt} = \phi - \frac{\beta SI}{N} - \mu S + \varphi R, \tag{11}$$

$$\frac{dE}{dt} = \frac{\beta SI}{N} - (\eta + \mu)E, \tag{12}$$

$$\frac{dI}{dt} = \eta E - (\sigma + \mu + \delta)I, \tag{13}$$

$$\frac{dT}{dt} = \sigma I - (\gamma + \mu)T, \tag{14}$$

$$\frac{dR}{dt} = \gamma T - (\varphi + \mu)R. \tag{15}$$

Table 1: TABLE OF PARAMETERS

S	Susceptible Individual
E	Exposed Individual
I	Infected population
T	Individual that received Treatment
R	Recovered population
ϕ	Recruitment rate
β	Transmission rate of infection
η	progression rate to the infectious class
σ	Treatment rate from infected to treated class
γ	Recovery rate under treatment
µ	Natural mortality rate
δ	Disease induced mortality rate
φ	immunity waning rate
N	Total host population

2.4 Model Value

Table 2: Model Parameters and Their Value

Parameter	Value
ϕ	0.029yr^{-1}
β	0.043yr^{-1}
η	0.002
σ	$0 \le \sigma \le 1$
γ	0.45yr^{-1}
μ	0.02yr^{-1}
δ	0.0003
φ	0.125

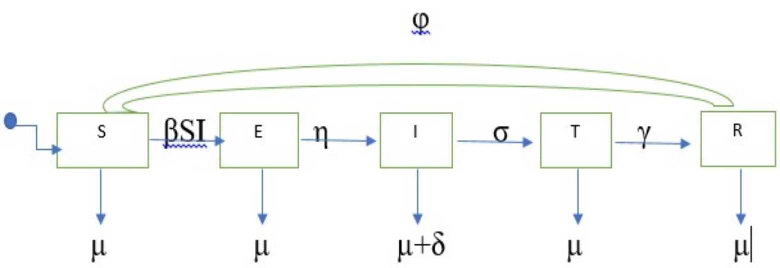

Figure 1: Schematic diagram of the proposed SEITR scabies model

2. 5 Existence and Uniqueness of The Model

Consider the scabies transmission model

$$\frac{d\varphi}{dt} = F(\varphi), \qquad \varphi(0) = \varphi_0,$$

with $\varphi = (S, E, I, T, R)^T$. Assume that F is continuous and its Jacobian matrix $JF(\varphi) = \left(\frac{\partial \varphi_i}{\partial x_j}\right)$ exists and is continuous in a region $\varphi \subset \mathrm{R}_+^5$ containing X_0. Then the model has a unique solution in φ for $t > 0$.

Proof. The continuity of the Jacobian matrix implies that F is locally Lipschitz in φ Hence, the classical existence–uniqueness theorem for ordinary differential equations guarantees a unique solution through X_0.

Proof;

From the model equation (11) – (15)

$$\frac{dS}{dt} = \phi - \frac{\beta SI}{N} - \mu S + \varphi R$$
$$\frac{dE}{dt} = \frac{\beta SI}{N} - \eta E - \mu E$$
$$\frac{dI}{dt} = \eta E - \sigma I - (\mu - \delta)I$$
$$\frac{dT}{dt} = \sigma I - \gamma T + \mu T$$
$$\frac{dR}{dt} = \gamma T - \varphi R - \mu R$$

Let;

$$L_1 = \varphi - \frac{\beta SI}{N} - \mu S + \phi R$$
$$L_2 = \frac{\beta SI}{N} - \eta E - \mu E$$
$$L_3 = \eta E - \sigma I - (\mu - \delta)I$$
$$L_4 = \sigma I - \gamma T + \mu T$$
$$L_5 = \gamma T - \phi R - \mu R$$

Then the following inequality holds; $\left|\frac{\partial L_i}{\partial(S,E,I,T,R)}\right| \le \varphi_i$

Where i = 1, 2...5, then system variables are required to obey Lipschitz continuity conditions such that the partial derivative exist

$$\varphi = \max\left(\left|\frac{\partial\varphi_1}{\partial S}\right|,\left|\frac{\partial\varphi_1}{\partial E}\right|,\left|\frac{\partial\varphi_1}{\partial I}\right|,\left|\frac{\partial\varphi_1}{\partial T}\right|,\left|\frac{\partial\varphi_1}{\partial R}\right|\right) = \left(\left|-\frac{\beta I}{N} - \mu\right|,|0|,\left|-\frac{\beta S}{N}\right|,|0|,|\phi|\right)$$

$$\varphi_2 = \max\left(\left|\frac{\partial\varphi_2}{\partial S}\right|,\left|\frac{\partial\varphi_2}{\partial E}\right|,\left|\frac{\partial\varphi_2}{\partial I}\right|,\left|\frac{\partial\varphi_2}{\partial T}\right|,\left|\frac{\partial\varphi_2}{\partial R}\right|\right) = \left(\left|\frac{\beta I}{N}\right|,|-\eta - \mu|,\left|\frac{\beta S}{N}\right|,|0|,|0|\right)$$

$$\varphi_3 = \max\left(\left|\frac{\partial\varphi_3}{\partial S}\right|,\left|\frac{\partial\varphi_3}{\partial E}\right|,\left|\frac{\partial\varphi_3}{\partial I}\right|,\left|\frac{\partial\varphi_3}{\partial T}\right|,\left|\frac{\partial\varphi_3}{\partial R}\right|\right) = \left(|0|,|\eta|,|-\sigma - (\mu - \delta|,|0|,|0|\right)$$

$$\varphi_4 = \max\left(\left|\frac{\partial\varphi_4}{\partial S}\right|,\left|\frac{\partial\varphi_4}{\partial E}\right|,\left|\frac{\partial\varphi_4}{\partial I}\right|,\left|\frac{\partial\varphi_4}{\partial T}\right|,\left|\frac{\partial\varphi_4}{\partial R}\right|\right) = \left(|0|,|0|,|\sigma|,|-\gamma-\mu|,|0|\right)$$

$$\varphi_5 = \max\left(\left|\frac{\partial\varphi_5}{\partial S}\right|,\left|\frac{\partial\varphi_5}{\partial E}\right|,\left|\frac{\partial\varphi_5}{\partial I}\right|,\left|\frac{\partial\varphi_5}{\partial T}\right|,\left|\frac{\partial\varphi_5}{\partial R}\right|\right) = \left(|0|,|0|,|0|,|\gamma|,|-(\varphi+\mu)|\right)$$

Since the partial derivatives of the right-hand side of the model system are continuous in the region φ, the associated vector field is locally Lipschitz continuous in φ. Therefore, by the Lipchitz condition, the model admits a unique local solution through every point in φ. Moreover, the positivity and boundedness results show that the solution remains in φ for all $t \geq 0$. Hence, the solution exists uniquely for all time $t \geq 0$.

2.6 Positivity of Solutions and Invariant Region

2.6.1 Positivity of Solutions

Theorem 2.2

Let $\Omega = \left\{(S, E, I, T, R) \in \mathrm{R}_+^5\right\}$.

If the initial data satisfy $S(0) \geq 0, \quad E(0) \geq 0, \quad I(0) \geq 0, \quad T(0) \geq 0, \quad R(0) \geq 0,$

then the susceptible component $S(t)$ remains nonnegative for all $t \geq 0$.

Consider the equation governing the susceptible population:

$$\frac{dS}{dt} = \phi - \frac{\beta SI}{N} - \mu S + \varphi R. \quad (16)$$

Removing the terms that do not contain S, we get

$$\frac{dS}{dt} \geq -\frac{\beta SI}{N} - \mu S.$$

Factoring out S, this becomes

$$\frac{dS}{dt} \geq -\left(\frac{\beta I}{N} + \mu\right)S.$$

Since

$$\frac{\beta I}{N} + \mu \geq 0,$$

therefore

$$\frac{dS}{S} \geq -\left(\frac{\beta I}{N} + \mu\right)dt. \quad (17)$$

Integrating both sides of (17) gives

$$\int \frac{dS}{S} \geq -\int\left(\frac{\beta I}{N}+\mu\right)dt.$$

Therefore

$$\ln S \geq -\left(\frac{\beta I}{N}+\mu\right)t + C. \tag{18}$$

Taking the exponential of both sides of (18)

$$S \geq e^{-\left(\frac{\beta I}{N}+\mu\right)dt+C}$$

Applying the initial condition $S(0)=S_0$, we obtain

$$S(t) \geq S(0)e^{-\left(\frac{\beta I}{N}+\mu\right)dt} \geq 0.$$

Therefore, $S(t)$ remains nonnegative for all $t \geq 0$.

$$S(t) \geq S(0)e^{-(\beta+\mu)t} \geq 0,$$

Similarly,

$$E(t) \geq E(0)e^{-(\eta+\mu)t} \geq 0, \qquad I(t) \geq I(0)e^{-(\sigma+\mu+\delta)t} \geq 0,$$
$$T(t) \geq T(0)e^{-(\gamma+\mu)t} \geq 0, \qquad R(t) \geq R(0)e^{-(\varphi+\mu)t} \geq 0.$$

Hence, all solutions remain nonnegative for all $t \geq 0$.

3.6.2 Invariant Region

To determine the invariant region for the model when $t \geq 0$, let

$$N(t) = S(t)+E(t)+I(t)+T(t)+R(t),$$

where $N(t)$ represents the total population at time t.

Then,

$$\frac{dN}{dt} = \frac{dS}{dt}+\frac{dE}{dt}+\frac{dI}{dt}+\frac{dT}{dt}+\frac{dR}{dt}.$$

Substituting the governing equations of the model into the above expression gives

$$\frac{dN}{dt} = \phi - \mu(S+E+I+T+R) - \delta I.$$

Since $N = S+E+I+T+R,$

Therefore

$$\frac{dN}{dt} = \phi - \mu N - \delta I.$$

Since $I(t) \geq 0$, we obtain

$$\frac{dN}{dt} \leq \phi - \mu N. \qquad (19)$$

Separating the variables and integrating (19) yields:

$$\int \frac{dN}{\phi - \mu N} \leq \int dt.$$

Hence,

$$-\frac{1}{\mu}\ln(\phi - \mu N) \leq t + C. \qquad (20)$$

By exponentiating both sides,

$$\phi - \mu N \leq Ke^{-\mu t},$$

for some positive constant K ,as $t \to \infty$, we have $e^{-\mu t} \to 0$, therefore

$$N(t) \leq \frac{\phi}{\mu}.$$

Thus, the biologically admissible region of the system is

$$\Omega = \left\{(S, E, I, T, R) \in \mathrm{R}_{+}^{5} : N(t) \leq \frac{\phi}{\mu}\right\}.$$

Therefore, all solutions of the model that start in Ω remain in Ω for all $t \geq 0$. Consequently, the region Ω is positively invariant for the scabies transmission model.

2.7 Disease Free Equilibrium

The disease free equilibrium (DFE) corresponds to a state where infection is absent from the population. Thus,

$$E^0 = I^0 = T^0 = R^0 = 0.$$

From the susceptible equation,

$$0 = \phi - \mu S^0.$$

Hence,

$$S^0 = \frac{\phi}{\mu}.$$

Therefore, the DFE becomes:

$$E_0 = \left(\frac{\phi}{\mu}, 0, 0, 0, 0\right). \tag{21}$$

2.8 Endemic Equilibrium

Let the endemic equilibrium be denoted by $E^* = (S^*, E^*, I^*, T^*, R^*)$,

where all state variables are nonnegative and at least one infected component is positive.

Setting the derivatives equal to zero gives

$$0 = \phi - \frac{\beta S^* I^*}{N^*} - \mu S^* + \varphi R^*, \tag{22}$$

$$0 = \frac{\beta S^* I^*}{N^*} - (\eta + \mu)E^*, \tag{23}$$

$$0 = \eta E^* - (\sigma + \mu + \delta)I^*, \tag{24}$$

$$0 = \sigma I^* - (\gamma + \mu)T^*, \tag{25}$$

$$0 = \gamma T^* - (\varphi + \mu)R^*. \tag{26}$$

Hence solving (22)-(26) yields:

$$E^* = \frac{\beta S^* I^*}{N^*(\eta + \mu)}, \quad I^* = \frac{\eta E^*}{\sigma + \mu + \delta}, \quad T^* = \frac{\sigma I^*}{\gamma + \mu}, \quad R^* = \frac{\gamma T^*}{\varphi + \mu}, \quad S^* = \frac{\phi + \varphi R^*}{\mu + \dfrac{\beta I^*}{N^*}}.$$

Thus, the endemic equilibrium exists implicitly as the solution of the above system.

2.9 Basic Reproduction Number

The basic reproduction number, denoted by R_0, is obtained by applying the next generation matrix method. We consider the infected compartments E and I.

Let $F = \begin{pmatrix} \dfrac{\beta SI}{N} \\ 0 \end{pmatrix}, \qquad V = \begin{pmatrix} (\eta + \mu)E \\ (\sigma + \mu + \delta)I - \eta E \end{pmatrix}.$

The Jacobian matrices at the DFE are

$$F = \begin{pmatrix} 0 & \dfrac{\beta S^0}{N^0} \\ 0 & 0 \end{pmatrix}, \tag{27}$$

and

$$V = \begin{pmatrix} \eta+\mu & 0 \\ -\eta & \sigma+\mu+\delta \end{pmatrix}. \tag{28}$$

Thus,

$$V^{-1} = \frac{1}{(\eta+\mu)(\sigma+\mu+\delta)} \begin{pmatrix} \sigma+\mu+\delta & 0 \\ \eta & \eta+\mu \end{pmatrix}. \tag{29}$$

Hence,

$$FV^{-1} = \frac{1}{(\eta+\mu)(\sigma+\mu+\delta)} \begin{pmatrix} \frac{\beta S^0 \eta}{N^0} & \frac{\beta S^0 (\eta+\mu)}{N^0} \\ 0 & 0 \end{pmatrix}. \tag{30}$$

Therefore, the spectral radius gives

$$R_0 = \frac{\beta S^0 \eta}{N^0 (\eta+\mu)(\sigma+\mu+\delta)}. \tag{31}$$

Since at the DFE,

$S^0 = N^0 = \frac{\phi}{\mu}$, this simplifies to

$$R_0 = \frac{\beta\eta}{(\eta+\mu)(\sigma+\mu+\delta)}. \tag{32}$$

2.10 Local Stability of the Disease-Free Equilibrium

From (11)-(15)

The Jacobian matrix of (11-15) gives:

$$J = \begin{pmatrix} -\mu & 0 & -\beta & 0 & \varphi \\ 0 & -(\eta+\mu) & \beta & 0 & 0 \\ 0 & \eta & -(\sigma+\mu+\delta) & 0 & 0 \\ 0 & 0 & \sigma & -(\gamma+\mu) & 0 \\ 0 & 0 & 0 & \gamma & -(\varphi+\mu) \end{pmatrix}. \tag{33}$$

The eigenvalues are obtained as;

$$\lambda_1 = -\mu, \quad \lambda_2 = -\gamma-\mu, \quad \lambda_3 = \varphi-\mu,$$
$$\lambda_4 = -\eta-\mu-\frac{1}{2}\delta+\frac{1}{2}\sqrt{\delta^2+4\eta\beta}, \quad \lambda_5 = -\eta-\mu-\frac{1}{2}\delta-\frac{1}{2}\sqrt{\delta^2+4\eta\beta}$$

The eigenvalues are negative, thus the system is locally asymptotically stable

2.11 Sensitivity Analysis

To measure how changes in a parameter affect (R_0), the normalized forward sensitivity index is defined as

$$\Upsilon_p^{R_0} = \frac{\partial R_0}{\partial \vartheta} \cdot \frac{\vartheta}{R_0}. \quad (34)$$

Using

$$R_0 = \frac{\beta\eta}{(\eta+\mu)(\sigma+\mu+\delta)},$$

the analytical sensitivity indices are:

$$\Upsilon_\beta^{R_0} = 1, \quad (35)$$

$$\Upsilon_\eta^{R_0} = \frac{\mu}{\eta+\mu}, \quad (35)$$

$$\Upsilon_\sigma^{R_0} = -\frac{\sigma}{\sigma+\mu+\delta}, \quad (36)$$

$$\Upsilon_\delta^{R_0} = -\frac{\delta}{\sigma+\mu+\delta}, \quad (37)$$

$$\Upsilon_\mu^{R_0} = -\frac{\mu}{\eta+\mu} - \frac{\mu}{\sigma+\mu+\delta}. \quad (38)$$

Table 3: Sensitivity indices of (R_0)

Parameter	Sensitivity Index
β	1
η	0.9090909091
σ	-0.9806805923
δ	-0.02942042
μ	-0.92942042

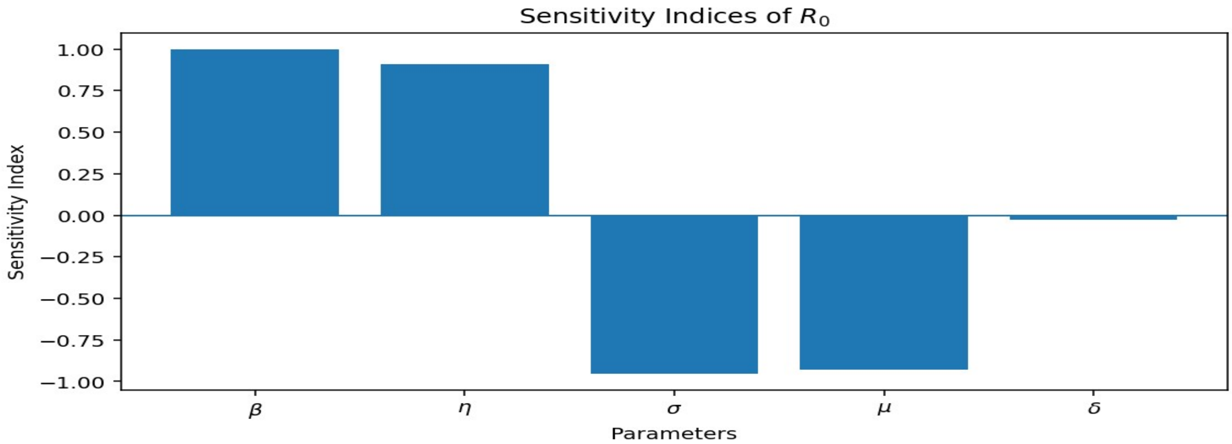

Figure 2: Sensitivity Indices of (R_0)

A positive sensitivity index implies that R_0 increases as the parameter increases, whereas a negative sensitivity index implies that R_0 decreases as the parameter increases. In particular, increasing the treatment rate σ reduces the reproduction number, which supports treatment as an important control strategy.

NUMERICAL STIMULATION

The numerical simulation of the proposed scabies model is based on the truncated HPM series solution. In this study, the first few terms of the HPM expansion are used to obtain approximate trajectories of the state variables.

$$\left.\begin{aligned}
&(1-w)\frac{dS(t)}{dt}+w\left(\frac{dS(t)}{dt}+\phi-\frac{\beta SI}{N}-\mu S(t)+\varphi R(t)\right)=0\\
&(1-w)\frac{dE(t)}{dt}+w\left(\frac{dE(t)}{dt}+\frac{\beta SI}{N}+(\eta-\mu)E(t)\right)=0\\
&(1-w)\frac{dI(t)}{dt}+w\left(\frac{dI(t)}{dt}+\eta E(t)-(\sigma+\mu-\delta)I(t)\right)=0\\
&(1-w)\frac{dT(t)}{dt}+w\left(\frac{dT(t)}{dt}+\sigma I(t)-(\gamma+\mu)T(t)\right)=0\\
&(1-w)\frac{dR(t)}{dt}+w\left(\frac{dR(t)}{dt}+\gamma T(t)-(\varphi+\mu)R(t)\right)=0
\end{aligned}\right\} \qquad (39)$$

Simplifying (39) yields:

$$\left.\begin{aligned}
&\frac{dS}{dt}-g\left(\phi-\frac{\beta SI}{N}-\mu S+\rho R\right)=0\\
&\frac{dE}{dt}-g\left(\frac{\beta SI}{N}-\eta E-\mu E\right)=0\\
&\frac{dI}{dt}-g\left(\eta E-\sigma I-(\mu-\delta)I\right)=0\\
&\frac{dT}{dt}-g\left(\sigma I-\gamma T+\mu T\right)=0\\
&\frac{dR}{dt}-g\left(\gamma T-\rho R-\mu R\right)=0
\end{aligned}\right\} \tag{40}$$

The approximate solution can be expressed as

$$S(t)=s_0+ws_1(t)+w^2s_2(t)+...+w^ns_n(t)$$

$$E(t)=e_0+we_1(t)+w^2e_2(t)+...+w^ne_n(t)$$

$$I(t)=g_0+wg_1(t)+w^2i_2(t)+...+w^ni_n(t)$$

$$T(t)=a_0+wt_1(t)+w^2t_2(t)+...+w^nt_n(t)$$

$$R(t)=r_0+wr_1(t)+w^2r_2(t)+...+w^nr_n(t)$$

Substituting and comparing the coefficients of

w, $w^0:\dot{s}_0(t)=0,\quad \dot{e}_0(t)=0,\quad \dot{i}_0(t)=0,\quad \dot{t}_0(t)=0,\quad \dot{r}_0(t)=0$

Solving yields

$$s_0(t)=s_o,\ e_0(t)=e_o,\ i_0(t)=i_o,\ t_0(t)=t_o,\ t_0(t)=t_o$$

Similarly, comparing the coefficients of w^1

$$\left.\begin{aligned}
\frac{dS_1(t)}{dt} &= \phi - \frac{\beta}{N}S_0(t)I_0(t) - \mu S_0(t) + \varphi R_0(t),\\
\frac{dE_1(t)}{dt} &= \frac{\beta}{N}S_0(t)I_0(t) + (\eta - \mu)E_0(t),\\
\frac{dI_1(t)}{dt} &= \eta E_0(t) - (\sigma + \mu - \delta)I_0(t),\\
\frac{dT_1(t)}{dt} &= \sigma I_0(t) - (\gamma + \mu)T_0(t),\\
\frac{dR_1(t)}{dt} &= \gamma T_0(t) - (\varphi + \mu)R_0(t)
\end{aligned}\right\} \tag{41}$$

Solving the system if equations result,

$$\left.\begin{aligned}
S_1(t) &= \left(\phi - \frac{\beta}{N}s_0 i_0 - \mu s_0 + \varphi r_0\right)t,\\
E_1(t) &= \left(\frac{\beta}{N}s_0 i_0 + (\eta - \mu)e_0\right)t,\\
I_1(t) &= \left(\eta e_0 - (\sigma + \mu - \delta)i_0\right)t,\\
T_1(t) &= \left(\sigma i_0 - (\gamma + \mu)t_0\right)t,\\
R_1(t) &= \left(\gamma t_0 - (\varphi + \mu)r_0\right)t
\end{aligned}\right\} \tag{42}$$

The coefficients of w^2 yields,

$$\left.\begin{aligned}
\frac{dS_2(t)}{dt} &= \phi - \frac{\beta}{N}S_1(t)I_1(t) - \mu S_1(t) + \varphi R_1(t),\\
\frac{dE_2(t)}{dt} &= \frac{\beta}{N}S_1(t)I_1(t) + (\eta - \mu)E_1(t),\\
\frac{dI_2(t)}{dt} &= \eta E_1(t) - (\sigma + \mu - \delta)I_1(t),\\
\frac{dT_2(t)}{dt} &= \sigma I_1(t) - (\gamma + \mu)T_1(t),\\
\frac{dR_2(t)}{dt} &= \gamma T_1(t) - (\varphi + \mu)R_1(t)
\end{aligned}\right\} \tag{43}$$

solving (43), to obtain the second approximations,

$$s_2(t)=\left[\frac{1}{2}-\frac{1}{N^2}t^2\begin{pmatrix}-\beta\eta N\sigma-\beta N\sigma\varphi\delta-\beta\varphi N+\beta^2\mu\sigma N-\beta\phi BN\\-\mu N^2\varphi+\mu^2N^2-\mu N^2\phi+\phi N^2\gamma-2\phi^2N^2\end{pmatrix}\right]$$

$$e_2(t)=\left[\frac{1}{N^2}t^2\begin{pmatrix}\beta\eta N-5\beta N\sigma-\beta\mu N+^2-4\mu\sigma+2\sigma\delta-2\gamma\sigma-\beta^2+2\beta\phi N+2N^2\eta^2-4N^2\eta\mu\\+2\mu^2N^2\end{pmatrix}\right]$$

$$i_2(t)=\left[-\frac{1}{N}t^2\left(-\eta\beta+\mu+5\phi\gamma-2\phi^2-4\phi\mu-\mu^2N+2N\mu\delta-2N\delta\eta-N\delta^2\right)\right]$$

$$t_2(t)=\left[t^2\left(4\sigma\eta-2\sigma^2-4\mu\sigma+2\sigma\delta-2\gamma\sigma+\gamma^2+2\gamma\mu+\mu^2\right)\right]$$

$$r_2(t)=\left[t^2\left(-10\gamma\sigma+5\gamma^2+\gamma\mu+5\phi\gamma-2\phi^2-4\phi\mu-2\mu^2\right)\right]$$

The solution in each compartment is represented as the sum of the computed approximation term such that:

$$S(t)=\sum_{\hbar=0}^{3}s_\hbar(t),\ E(t)=\sum_{\hbar=0}^{3}e_\hbar(t),\ I(t)=\sum_{\hbar=0}^{3}i_\hbar(t),\ T(t)=\sum_{\hbar=0}^{3}t_\hbar(t),\ R(t)=\sum_{\hbar=0}^{3}r_\hbar(t)$$

3.0 RESULTS

The result of the semi analytical methods of the HPM is obtained:

$$S(t)=500-9.8336374t+2.785446075t^2-0.297294640625t^3,$$

$$E(t)=200-2.0373626t-0.314698708t^2+0.039011959075t^3,$$

$$I(t)=100-26.57t+3.580927137t^2-0.32213515372t^3,$$

$$T(t)=90-13.7t-0.37575t^2+0.352268090625t^3,$$

$$R(t)=20+37.6t-5.8085t^2+0.2243817t^3.$$

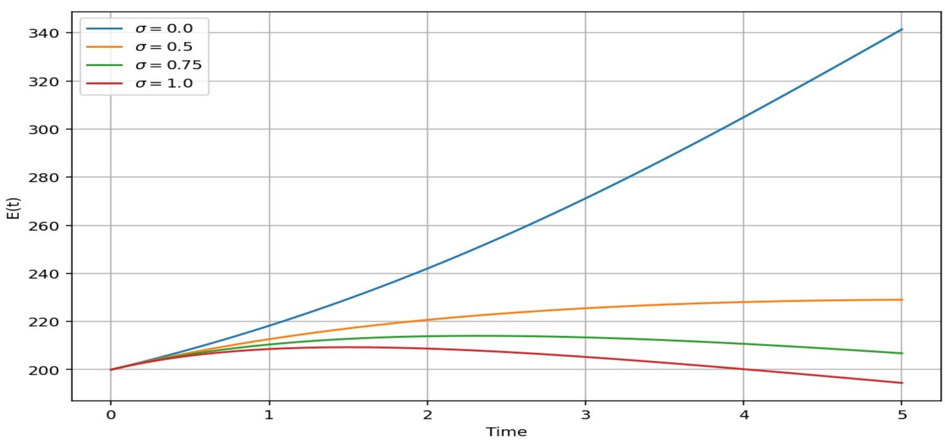

Figure 1: Dynamics of the exposed population at different treatment rates

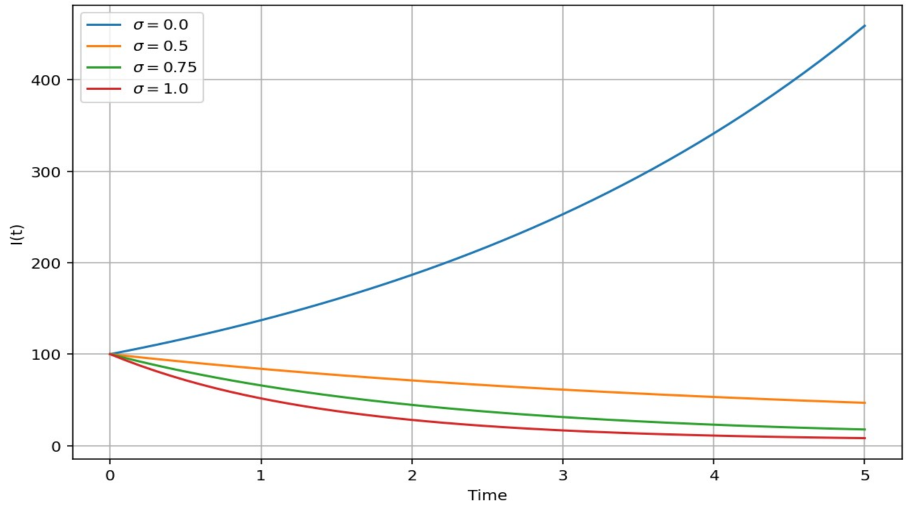

Figure 2: Effect of treatment rate on the infected population

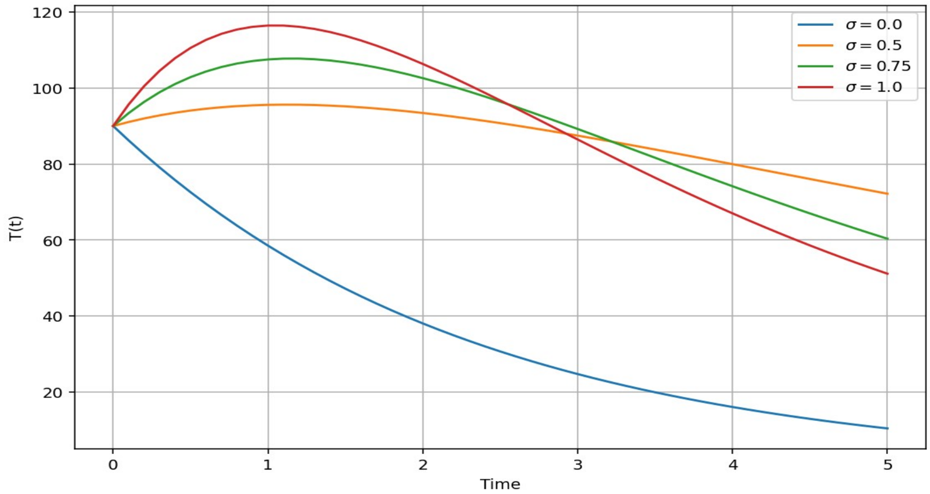

Figure 3: Dynamics of the treated population at different treatment rates

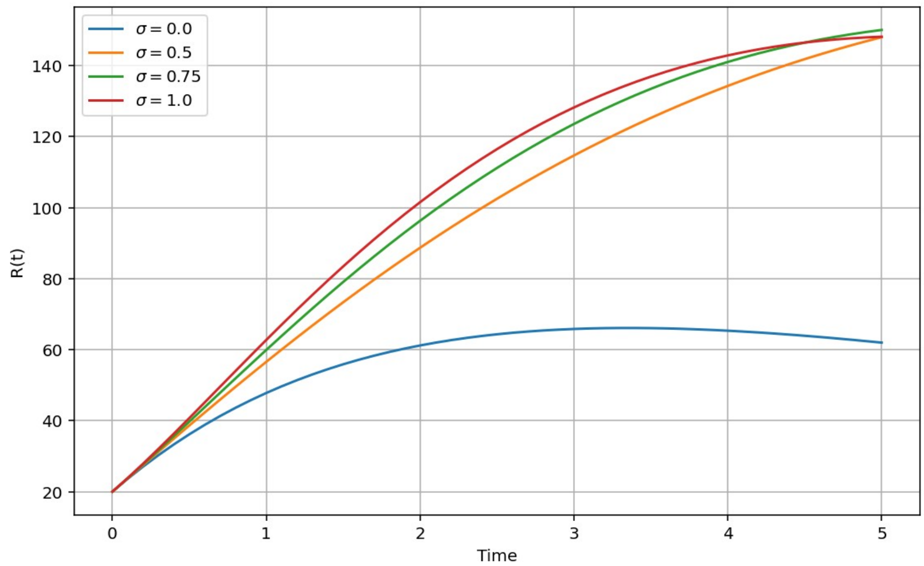

Figure 4: Response of the recovered population to changes in treatment rate

4. DISCUSSION

The analytical and numerical results consistently indicate that treatment plays a crucial role in the control of scabies transmission. From the analytical point of view, the treatment rate (σ) was identified as one of the most influential parameters affecting the basic reproduction number, and its negative sensitivity index shows that increasing treatment lowers the transmission potential of the disease. This theoretical observation is supported by the numerical simulations presented in Figures 1–4, where higher values of (σ) lead to reductions in the exposed and infected populations, a temporary increase in the treated population, and a substantial rise in recovery.

Figure 1 illustrates the influence of the treatment rate (σ) on the exposed population $E(t)$. In the absence of treatment $(\sigma = 0)$, the exposed class increases steadily throughout the simulation period, reflecting sustained transmission and the continuous movement of susceptible individuals into the exposed compartment. When treatment is introduced at $(\sigma = 0.5)$, the exposed population still rises, but at a slower rate. For higher treatment levels ($\sigma = 0.75$ and $\sigma = 1$), the exposed class shows only a slight initial increase before declining over time. This behaviour suggests that improved treatment reduces the number of infectious individuals capable of generating new exposures, thereby weakening the force of infection. Hence, the trend observed in Figure 1 is consistent with the analytical result that stronger treatment suppresses transmission.

The treatment effect is seen in Figure 2 for the infected population $I(t)$. When no treatment is administered $(\sigma = 0)$, the infected class rises rapidly, indicating persistent transmission and continued spread of the disease within the population. However, for all positive values of σ, the infected population declines with time, and this decline becomes steeper as σ increases. The most rapid decrease occurs when $\sigma = 1$, where the infected class reaches its lowest level over the simulation period. This pattern agrees with the sensitivity analysis, which showed that higher treatment reduces the basic reproduction number. Epidemiologically, this means that treatment shortens the infectious period and limits the number of secondary infections generated by infected individuals.

The behaviour of the treated population $T(t)$ is presented in Figure 3. When treatment is absent $(\sigma = 0)$, the treated class decreases continuously because no infected individuals enter treatment. Once treatment is introduced, however, the treated population rises initially, reaches a peak, and then gradually declines. The early increase becomes more evident as σ increases, reflecting the higher movement of infected individuals into the treatment class at greater treatment rates. The later decline occurs because the infectious pool is progressively reduced, leaving fewer individuals available to move into treatment. This transient behaviour highlights the intermediate role of treatment in transferring individuals from active infection to recovery.

Figure 4 shows the corresponding effect of treatment on the recovered population $R(t)$. In the absence of treatment $(\sigma = 0)$, the recovered class increases only slightly and eventually approaches a much lower level than in the treated cases. For positive values of σ, recovery accumulates more rapidly and attains considerably higher levels over time. In particular, the curves for $\sigma = 0.75$ and $\sigma = 1.0$ produce the largest increase in the recovered population, indicating that stronger treatment enhances progression to recovery and contributes significantly to disease control. This result is fully in line with the model structure, since treatment moves individuals from the infected class to the treated class, from which they eventually recover.

As observed, the results in Figures 1–4 show that increasing the treatment rate σ has a beneficial effect on the dynamics of scabies transmission. Analytically, treatment reduces the basic reproduction number and strengthens the threshold condition necessary for disease control. Numerically, it decreases the exposed and infected populations, produces the expected temporary rise in the treated population, and substantially increases the recovered population. These findings demonstrate that improving treatment coverage and effectiveness is essential for reducing transmission, limiting disease persistence, and strengthening scabies control at the population level.

5. CONCLUSION

i. The sensitivity results identify the treatment rate, together with the transmission rate, as one of the dominant factors governing the basic reproduction number, indicating that improved treatment reduces the transmission potential of the disease.

ii. Increasing the treatment rate produces substantial reductions in the exposed and infected populations over time, showing that stronger treatment weakens the force of infection and limits disease persistence within the population.

iii. The treated population rises initially as more infected individuals enter treatment and later declines as the infectious reservoir is depleted, while the recovered population increases markedly with higher treatment intensity, confirming that treatment enhances progression to recovery.

iv. Increased treatment coverage and effectiveness therefore provide an important population-level mechanism for reducing prevalence and improving the prospects for sustained scabies control.

Future research may extend the present model by incorporating reinfection, treatment non-compliance, variation in treatment efficacy, and heterogeneous contact structure. Household-based and spatial transmission pathways may also be included to reflect the close-contact nature of scabies spread. In addition, calibration with epidemiological data would improve the predictive performance of the model and strengthen its usefulness for public health planning and intervention design.

References

Alaje, A. I., Olayiwola, M. O., & Odeleye, J. F. (2025). Spatiotemporal analysis of Zika virus transmission dynamics incorporating human mobility and seasonal variations using modified homotopy perturbation method. *Journal of Umm Al-Qura University for Applied Sciences, 11*, 491–508.

Ayoola, T. A., Alaje, A. I., Muhammad, H., Adekunle, T. A., Popoola, A. O., & Bashiru, K. A. (2025). *Forecasting the trend of COVID-19 under imperfect vaccination using the homotopy perturbation method* [Preprint]. *Research Square*.

Centers for Disease Control and Prevention. (2023a, December 18). *Clinical care of scabies*.

Centers for Disease Control and Prevention. (2023b, December 18). *Preventing scabies*.

Centers for Disease Control and Prevention. (2024a, September 9). *Treatment of scabies*. U.S. Department of Health and Human Services.

Centers for Disease Control and Prevention. (2024b, February 23). *How scabies spreads*.

Fadhal, E., Raza, A., Rocha, E. M., Alfwzan, W. F., Rafiq, M., Ahmed, N., & Bilal, M. (2025). Dynamical analysis of scabies delayed epidemic model with second-order global stability. *PLOS ONE, 20*(4), e0319095.

He, J.-H. (1999). Homotopy perturbation technique. *Computer Methods in Applied Mechanics and Engineering, 178*(3–4), 257–262.

Hethcote, H. W. (2000). The mathematics of infectious diseases. *SIAM Review, 42*(4), 599–653.

Iyengar, L., Chong, A. H., & Steer, A. C. (2024). Scabies: A clinical update. *Medical Journal of Australia, 221*(10), 558–563.

Kermack, W. O., & McKendrick, A. G. (1927). A contribution to the mathematical theory of epidemics. *Proceedings of the Royal Society of London. Series A, Containing Papers of a Mathematical and Physical Character, 115*(772), 700–721.

Lydeamore, M. J., Campbell, P. T., Regan, D. G., Tong, S. Y. C., Andrews, R. M., Steer, A. C., Romani, L., Kaldor, J. M., McVernon, J., & McCaw, J. M. (2019). A biological model of scabies infection dynamics and treatment informs mass drug administration strategies to increase the likelihood of elimination. *Mathematical Biosciences, 309*, 163–173.

Mitchell, E. M., Wallace, M. W., Marshall, J. M., Whitfeld, M., & Romani, L. (2024). Scabies: Current knowledge and future directions. *Frontiers in Tropical Diseases, 5*, Article 1429266.

Olaosebikan, M. L., Kolawole, M. K., Alaje, A. I., Oladapo, A. O., & Adediran, I. A. (2025). Mathematical modelling of hepatitis B virus dynamics evaluating the role of vaccination and metabolic factors in disease progression. *Quality & Quantity*. Advance online publication.

Roy, T., & Maiti, D. K. (2023). An optimal and modified homotopy perturbation method for strongly nonlinear differential equations. *Nonlinear Dynamics, 111*, 15215–15231.

Uzun, S., Durdu, M., Yürekli, A., Mülayim, M. K., Akyol, M., Velipaşaoğlu, S., Harman, M., et al. (2024). Clinical practice guidelines for the diagnosis and treatment of scabies. *International Journal of Dermatology, 63*(12), 1642–1656.

van der Linden, N., van Gool, K., Gardner, K., Dickinson, H., Agostino, J., Regan, D. G., Dowden, M., & Viney, R. (2019). A systematic review of scabies transmission models and data to evaluate the cost-effectiveness of scabies interventions. *PLOS Neglected Tropical Diseases, 13*(3), e0007182.

World Health Organization. (2023, May 31). *Scabies*.